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MODULE 20
INDUSTRIAL HEAT EXPOSURE

Presenter: Capt. Mario E. Fajardo, M.D.
Occupational Medicine Physician
Human Factors Division (G-WKS-3)

Disclosure: Capt. Fajardo does not have any financial arrangements or affiliations with any corporate organizations that might constitute a conflict of interest with regard to this continuing education activity.

Goals:

1. Learn about body temperature regulation.
2. Know the effects heat stress on worker performance.
3. Understand the major factors in heat tolerance.
4. Learn the medical aspects of heat stress
5. Learn about prevention of heat illness.

Note: This lecture material is a summary of information extracted from various sources dealing with this extensive subject matter. The presenter takes no authorship of this material. For more detailed information readers are referred to the following reference material:

1. Textbook of Clinical Occupational and Environmental Medicine, Rosentock/Cullen, Chapter 24-Thermal Stressors, Ethan Nadle/Mark R. Cullen.
2. The Industrial Environment-its Evaluation and Control (NIOSH)- Chapter 30-Physiology of Heat Stress, David Minard, M.D., Ph.D.
3. Health Effect of Toxic Substances, Second Edition, M.J. Malachowski, PhD, Chapter 15- Medical Monitoring Treatment and Management.

Additional Material:
At the end of the lecture additional material will be presented on the following subjects:

1. Physiological Adaptation to Heat Stress.
2. Prevention of Heat Illness.
3. Management of Heat Illness.

INDUSTRIAL HEAT EXPOSURE

Heat exposure is one of the oldest recognized occupational hazards. Despite climate control conditions implemented in most developed countries and the technological advances of the 21st Century, problems relating to heat stress in the workplace remain a widespread problem. Heat exposure outside the work setting, particularly for the elderly and the very young, also remains a particular problem. Reliable incidence and prevalence data on environmental exposures have not been available in most parts of the world, however, where records and data are available, the effects of heat as well as cold, are noted to be a significant contributor in many compensation and hospitalization cases. Heat generated by the use of heavy machinery, as well as the metabolic heat produced by workers in the performance of their jobs results in levels of industrial environmental heal which often exceeds the level of heat exposure encountered in the natural environment. Heat generated at the work site at levels sufficient to interfere with normal function is commonly the result of three factors:

1) Heat generated by the individual over time,
2) External heat load, and
3) Ability to dissipate heat

The amount of *heat generated by workers* relates to the intensity and type of work being performed; *the heat load* is determined by the ambient temperature plus radiant temperature from other sources such as heat elements, sunlight and lamps which add to the load and; the *ability to eliminate heat* is determined by clothing and environmental conditions. For example, environmental conditions such as low humidity and high air movement speed heat removal from the skin, while water repellent clothing, including personal protective equipment (PPE), tends to impede heat dissipation. PPE (in particular respirators) utilized by HAZMAT teams and search and rescue personnel, tends to impede the elimination of heat. It is well known that the level of heat stress increases when protective equipment, space confinements, impermeable clothing, or any other devices hamper the ability of the body to cool itself and the individual's physiologic capacity to regulate body temperature is exceeded. In order to prevent a loss in cooling capacity one must either lower the physical work demands or lower the environmental heat. Individuals subject to heat stress should be initially evaluated for the ability to work in protective equipment. Medical providers should make the following notations:

1. Ability to perform assigned tasks based on medical history or physical exam.
2. Ability to wear protective equipment and/or ability to meet job specifications.
3. Limitations on the use of personal protective equipment.
4. Make a work determination based on the individual's profile.

Many outbreaks of heat related illness have occurred in selective high-work settings such as military training environments that were not particularly hot. The provider should pay close attention to the level of physical activity and intensity of work being performed and not solely to environmental factors. Remember that increased physical activity adds heat to the body at a greater rate than a hot environment. The body will not see an increase in core temperature unless the air and/or radiant temperatures are higher than the body temperature. However, work activities can produce 80-700 kcal/hour and sun exposure can add an additional 150 kcal/hour. Since each kcal of heat per kilogram of body weight can raise the body temperature by 0.8^0C, if heat loss remains constant, the risk for hyperthermia for those engaged in physical activities in the heat is considerable. Evidence of heat stress may initially manifest with poor or impaired performance and/or with clinical signs of heat illness. In order to properly assess the presence of heat illness one must have knowledge of several factors:

1. Ambient temperature
2. Radiant temperature
3. Relative humidity
4. Air velocity
5. Physical activity level
6. Clothing cover and permeability

Several of the aforementioned factors can be measured independently or collectively, using the wet-bulb globe thermometer. It should be noted, however, that the wet-bulb globe index and corrected effective-temperature tend to correlate better with the level of heat stress than using temperature alone.

PHYSIOLOGY OF HEAT STRESS

The body's response to a heat increase is to unload the excess and maintain homeostasis. Man regulates body temperature by physiologic control of blood flow from sites of heat production (muscles; deep tissues) to the cooler body surface where heat is dissipated through physical channels- radiation; convection; and evaporation. Despite variations in the thermal environment, the body seeks to maintain homeostasis. This is accomplished by distributing heat throughout the body by convection in the blood stream. Thermal sensors within the brain also respond to the increased body temperature by increasing nervous system activity, which directs other heat dissipation responses- increased blood flow to the skin and sweating. The resultant effect is an increased rate of transfer of heat from the body core to the environment. Homeostasis is achieved when the rate of heat loss equals the rate of heat production. Under ambient heat and cold conditions superficial tissues (skin, subcutaneous fat) reflect mean temperature changes on the skin surface more widely than the warm body core.

The energy required by the body to meet its life-sustaining functions is produced by an enzymatically controlled oxidative combustion of fuel substrates (carbohydrates, fat, protein). The end products of these reactions are CO_2, water and nitrogen wastes. The heat produced by the body as a result of these reactions equals the heat produced when these substances are oxidized at high temperatures outside the body- *exothermic reactions*. These exothermic reactions form the basis for indirect calorimetry; in which metabolically produced heat can be measured by the rate of oxygen uptake during rest or activity- one liter of O^2 being equivalent to 5kcal of heat output. Metabolic heat production can be obtained, in a clinical laboratory setting, by measuring the difference in O^2 concentration between inspired and expired air. At metabolic rest individual differences in the amount of heat production per unit area are insignificant. However, body size, age, physical fitness and muscular development play a significant role in the wide differences found when determining the maximum capacity of work performance. The maximum rate of oxygen uptake (VO^2 max) during a brief strenuous work effort is an important measure of work capacity. The VO^2 max among healthy workers varies between 2.0 and 4.0 liters/min.

The “temperature-regulating center” located in the hypothalamus responds to increases in its own temperature as well as to incoming (afferent) nerve impulses from “warm” skin receptors. In response to these impulses it then activates heat loss through increased blood flow and sweating. Under comfortable ambient conditions approximately 25% of metabolic heat production is transferred from the skin to the air by convection; another 50% is lost through radiative transfer to other surfaces; and the remaining 25% is lost via evaporation or by warming inspired air. Respiratory heat loss, which accounts for 8-10% of the resting metabolic heat production, plays little role in temperature regulation. The subjective sensation of heat or warmth is dependent on skin temperature. However, the sensation of *heat discomfort* is based not only on heat sensation but also on the level of physiological strain. Thermal comfort scales are available which help determine the limits of ambient temperatures, activity levels, and clothing under which heat balance can be achieved without inducing thermal strain. In well-acclimatized individuals the estimated 2.5 million eccrine glands can secrete sweat, under maximum central drive, at peak rates of more than 3 kg/hr for at least 1 hour and can maintain a level of 1-1.5 kg/hr for several more hours. Interestingly, the central drive for sweating is determined by the metabolic work rate but the actual sweat output is modulated by skin temperature to meet evaporative requirements.

A rise in body core temperature greater than 2 - 2.5^0C leads to a progressive redistribution of blood flow (increased blood volume to the skin) with side effects, which are manifested by a reduction in work performance and ability for, prolonged activity. The reduction in central circulating blood volume reduces the filling pressure of the heart and thus the ability of the heart to maintain adequate

blood flow. Unless compensatory reflexes are activated the arterial blood pressure and cerebral blood flow fall with resultant clinical manifestations of lightheadedness, dizziness, disorientation, and finally loss of consciousness. Compensatory mechanisms to a fall in filling pressure include a rise in heart rate and constriction of non-essential vascular beds, including the skin. The goal is to redistribute blood from the peripheral areas to the body's core in order to maintain an effective central blood volume.

The following table indicates the recommended work duration for a fit and acclimatized worker:

Adjusted temperature Centigrade	Adjusted temperature Fahrenheit	Normal work ensemble Minutes of work	Impermeable ensemble Minutes of work
32.2	90 or more	45	15
30.8 - 32.2	87.5 - 90	60	30
26.1 – 30.8	82.5 – 87.5	90	60
25.3 – 26.1	77.5 – 82.5	120	90
22.5 – 25.3	72.5 – 77.5	150	120

Conditions or events limiting the normal thermoregulatory mechanism will cause the body core temperature to rise precipitously. Predisposing or common high-risk factors with the potential of affecting the thermoregulatory center include cardiovascular diseases, alcoholism, diabetes, infections, fever, dehydration, obesity, cystic fibrosis, scleroderma, prior history of heat stroke and hyperthyroidism. Therapeutic and recreational drugs have also been implicated as prevalent risk factors, as is any agent that interferes with autonomic function, vascular tone, motor activity or cardiovascular response. Changes in core to surface gradient alter the rate of blood flow from the warm core to the cooler body surface to meet needs in heat conductance.

A rise in body core temperature leads to a number of pathological changes including the inhibition of spermatogenesis, development of hepatic insufficiency, pancreatitis and disseminated intravascular coagulation resulting from fibrinolysis and activation of platelet activity. Rhabdomyolysis and myocardial necrosis have also been reported as a result of myocardial and skeletal muscle disruption; renal function and central nervous system activities are eventually affected as a result of the cascade of other body dysfunctions. Many of these conditions, however, can be alleviated through a process of acclimatization, which can be done through gradual increased exposures to both heat stress and exercise stress. Maximum acclimatization can be achieved after a period of about two weeks of regular exposure. Heat acclimatization is characterized by the following parameters: improved cardiovascular fitness; alterations in sweating and; enhanced renal function. The net effect of acclimatization is a markedly increased heat stress tolerance.

The following table illustrates the amount of heat generated by some common occupational activities:

Activity	Rate (kcal/hr)
Bicycling	360-600
Climbing stairs	360-720
Construction tasks	300-600
Domestic work	75-300
Driving	120-180
Heavy labor/factory work	210-400
Light assembly/inspection	90-160
Machine operator/manual trade	120-240
Office work (sitting)	75-120
Resting (sitting or lying)	60-90
Standing (inactive)	90-120
Swimming	300-880
Walking on flat surface (3-5 km/hr)	300-400

HEAT STRESS DISORDERS

Conditions associated with heat stress can be classified into acute and subacute disorders. It should also be noted that heat exposure is responsible for a significant number of skin related conditions. Among the conditions associated with heat exposure, the following will be discussed in some detail:

Acute-	Subacute-	Skin Disorders
Heat syncope	Infertility	Erythema ab igne
Heat edema	Teratogenesis	Miliaria
Heat tetany		
Heat cramps		
Heat exhaustion		
Heat stroke		

Heat syncope- this relatively benign condition is indistinguishable from syncope resulting from any other cause. It commonly affects an unacclimatized individual as a result of vasodilatation and diminished venous return during the early adaptation to heat stress. Those under the influence of medications such as diuretics, who are dehydrated or whose autonomic function is impeded are at greatest risk. Presenting symptoms usually are dizziness or LOC after heat exertion. Clinically there is associated tachycardia, postural hypotension, normal

temperature and sweating. These patients are usually managed by placing them at rest in a recumbent position and providing salt and water.

Heat edema- this condition typically occurs a couple of days after sun exposure. It is considered a second physiological adaptation to heat stress by an unacclimatized individual. It is believed to be secondary to a relative oliguria and hyperaldosteronism. The diagnosis of this self-limiting process is confirmed by the history and physical examination. It requires no medical intervention.

Heat tetany- this occurs in an unacclimatized individual due to hyperventilation and subsequent systemic alkalemia. Perioral paresthesias followed by frank tetany are the presenting complaints. The only management modality is removal from the heat.

Heat cramps- cramping of muscles due to heat stress tend to occur in acclimatized individuals who perform strenuous physical activity under a high degree of heat stress. The cramps commonly occur late in the day and tend to involve the muscle groups most strenuously exercised. Correcting the underlying hyponatremia, by administering large volumes of fluids with adequate sodium concentration is key to resolving the problem.

Heat Exhaustion- this is a manifestation of adequate fluid replacement with poor salt replacement. This syndrome manifests with nausea, vomiting, diarrhea, and generalized weakness. The individual may present pale and clammy with loss of skin elasticity, normal body temperature and renal function, hypotension and tachycardia and the hallmark of hyponatremia. Additional fluid replacement with adequate correction of the hyponatremia and moving the individual to a cool place is essential.

Heat stroke- this represents the ultimate failure of the heat dissipating mechanisms to compensate for heat stress. It results in a sudden rise in body-core temperature to over 40^0C with resulting profound central nervous system dysfunction manifested by altering levels of consciousness, delirium and psychotic behavior. This may be accompanied by hypotension and circulatory collapse and pulmonary edema. Laboratory parameters commonly reveal abnormalities in the creatinine phosphokinase, serum potassium, uric acid hepatic enzymes and phosphate levels. It is important to remember that the clinical presentation will vary according to the level and severity of the heart exposure. A team of experienced intensivists commonly carries out management of heat stroke. The complexity of the renal, cardiovascular, neurologic metabolic and hematological disturbances cannot be successfully conducted in a general clinical setting.

Reproductive effects- animal data not yet verified by well-controlled human studies indicate that thermal stress to the developing fetus may have some

teratogenic effects in humans. Additionally, quantitative studies indicate a 20% drop in baseline sperm levels in summer months when compared to cooler ones.

PREVENTION

Prevention of heat stress involves a combination of environmental and administrative measures. Whenever possible steps should be taken to control the temperature, humidity, air velocity and radiation sources of the spaces where strenuous physical activity is to occur. The use of rapid air circulation systems, de-humidifiers, and/or shielding of heat sources should be implemented in any heat stressful operation. Administrative controls can also be implemented when manipulation of climate conditions becomes unfeasible, such as working outdoors. Establishing acclimatization programs, arranging work schedules and providing adequate amounts of fluid and salt replacement are notable implementations of heat-stress reduction practices.

PROCEED TO POST-TEST & ADDITIONAL READING MATERIAL